Amendments to the Claims:

Please **cancel** claims 1-36 without prejudice to or disclaimer of the underlying subject matter, and please **add** the following claims 37-76:

1-36. (Cancelled)

37. (New) A compound which has binding affinity for a tumor-specific molecule and is able to effect dyslocalization of the tumor-specific molecule.

38. (New) The compound of claim 37, in which the dyslocalization inhibits the growth of tumor-specific cells.

39. (New) The compound of claim 37, in which the dyslocalization induces apoptosis in tumor-specific cells.

40. (New) The compound of claim 37, which is a peptide, oligopeptide, protein, fusion protein, or an organic molecule having a molecular weight of < 5000, < 1000 or < 500.

41. (New) The compound of claim 37, in which the tumor-specific molecule is a peptide, oligopeptide, protein, fusion protein, RNA or DNA.

42. (New) The compound of claim 37, which has a binding affinity of 10^{-5} to 10^{-12}.

43. (New) The compound of claim 37, which has a binding affinity of 10^{-7} to 10^{-9}.

44. (New) The compound of claim 37, in which the tumor-specific molecule is not present in healthy cells or is present in another form.

45. (New) The compound of claim 37, in which the tumor-specific molecule is a fusion protein.

46. (New) The compound of claim 37, in which the tumor-specific molecule is AML1-ETO.

47. (New) The compound of claim 37, in which the tumor-specific molecule has a DNA binding domain, a signal peptide, kinase activity, chromatin-modulatory properties, protein-protein interaction domains or transcriptional properties.

48. (New) The compound of claim 37, in which the dyslocalization binds the tumor-specific molecule to a nucleic acid sequence which regulates the transcription of a gene.

49. (New) The compound of claim 37, in which the dyslocalization binds the tumor-specific molecule to a nucleic acid sequence which regulates the transcription of a gene, thereby activating or inhibiting the transcription of the gene.

50. (New) The compound of claim 37, in which the compound comprises the peptide sequence of the c-myb DNA binding domain.

51. (New) The compound of claim 37, in which the compound comprises the peptide sequence of the AML-1 binding domain of the myeloid elf like factor.

52. (New) The compound of claim 37, in which the compound comprises the peptide sequence of the c-myb DNA binding domain and the peptide sequence of the AML-1 binding domain of the myeloid elf like factor.

53. (New) The compound of claim 37, in which the compound has the sequence shown in SEQ ID NO: 1.

54. (New) A nucleic acid encoding a peptide or protein of claim 53.

55. (New) The nucleic acid of claim 54, which is a DNA or RNA.

56. (New) A vector comprising a nucleic acid of claim 54.

57. (New) A host cell having a vector of claim 56.

58. (New) A medicament comprising a compound of claim 37, a nucleic acid of claim 54, a vector of claim 56, or a host cell of claim 57.

59. (New) The medicament of claim 58, which further comprises a pharmaceutically acceptable carrier.

60. (New) The medicament of claim 58, which is formulated for oral, intravenous or intramuscular administration.

61. (New) A method of treating tumors comprising administering to a patient in need thereof a compound of claim 37, a nucleic acid of claim 54, a vector of claim 56, or a host cell of claim 57.

62. (New) The method of claim 61, wherein the tumor is leukemia.

63. (New) The method of claim 61, wherein the tumor is acute myeloid leukemia.

64. (New) A method for the preparation of a compound of claim 37, in which the peptide or protein is recombinantly expressed or obtained by protein synthesis.

65. (New) A method for identifying a compound suitable for the treatment of tumors, in which:

(a) a tumor-specific molecule is identified;

(b) a compound which has a binding affinity for said tumor-specific molecule and is able to effect a dyslocalization of said tumor-specific molecule is identified.

66. (New) The method of claim 65, in which compounds are identified, in which the dyslocalization inhibits the growth of tumor-specific cells or induces apoptosis in tumor-specific cells.

67. (New) The method of claim 65, in which the tumor-specific molecule is identified by microarray analyses, 2D protein gel electrophoreses with subsequent identification by mass spectrometry, or a combination of said methods.

68. (New) The method of claim 65, in which the compound which has a binding affinity for the tumor-specific molecule and is able to effect a dyslocalization of the tumor-specific molecule is a protein, an RNA, a DNA or an organic compound.

69. (New) The method of claim 65, in which the compound which has a binding affinity for the tumor-specific molecule and is able to effect a dyslocalization of the tumor-specific molecule is identified by means of high-throughput screening methods.

70. (New) The method of claim 65, in which the compound which has a binding affinity for the tumor-specific molecule and is able to effect a dyslocalization of the tumor-specific molecule has been constructed from two parts.

71. (New) The method of claim 70, in which one part of the compound has a binding affinity for the tumor-specific molecule, and the second part is able to effect the dyslocalization of the tumor-specific molecule.

72. (New) The method of claim 70, in which the two parts are identified in separate screening methods.

73. (New) A method for the preparation of a medicament, comprising the steps of:

(a) identifying a compound suitable for the treatment of tumors by a method of claim 64;

(b) preparing the compound by synthesis or recombinantly; and

(c) formulating the compound to give a medicament.

74. (New) The method of claim 73, wherein the medicament is suitable for the treatment of tumors.

75. (New) The method of claim 73, wherein the medicament is suitable for the treatment of leukemia.

76. (New) The method of claim 73, wherein the medicament is suitable for the treatment of acute myeloid leukemia.